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 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
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 NEWS  3  May 10   PROUSDDR now available on STN
 NEWS  4  May 19   PROUSDDR: One FREE connect hour, per account, in both May
                   and June 2004
 NEWS  5  May 12   EXTEND option available in structure searching
 NEWS  6  May 12   Polymer links for the POLYLINK command completed in REGISTRY
 NEWS  7  May 17   FRFULL now available on STN
 NEWS  8  May 27   New UPM (Update Code Maximum) field for more efficient patent
                   SDIs in CAplus
 NEWS  9  May 27   CAplus super roles and document types searchable in REGISTRY
 NEWS 10  May 27   Explore APOLLIT with free connect time in June 2004
 NEWS 11  Jun 22   STN Patent Forums to be held July 19-22, 2004
 NEWS 12  Jun 28   Additional enzyme-catalyzed reactions added to CASREACT
 NEWS 13  Jun 28   ANTE, AQUALINE, BIOENG, CIVILENG, ENVIROENG, MECHENG,
                   and WATER from CSA now available on STN(R)

 NEWS EXPRESS  MARCH 31 CURRENT WINDOWS VERSION IS V7.00A, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 26 APRIL 2004
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=> file medline, uspatful, dgene, embase, wpids, fsta, biosis, hcaplus
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                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   0.42       0.42

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FILE 'USPATFULL' ENTERED AT 18:02:51 ON 30 JUN 2004
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=> s albumin fusion protein?
   3 FILES SEARCHED...
L1         8757 ALBUMIN FUSION PROTEIN?

=> s brain derived neurotrophic factor protein?
   3 FILES SEARCHED...
L2          135 BRAIN DERIVED NEUROTROPHIC FACTOR PROTEIN?

=> s s l1 and l2
MISSING OPERATOR S L1
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l1 and l2
L3            0 L1 AND L2

=> s l2 and fusion
L4            1 L2 AND FUSION

=> d l4 ti abs ibib tot

L4   ANSWER 1 OF 1  USPATFULL on STN
TI     Cystine knot growth factor mutants
AB     Compositions and methods based on mutant Cystine Knot Growth Factors
       (CKGFs) comprising amino acid substitutions relative to the wild type
       hormone/growth factor. Mutated glycoprotein hormones, including thyroid
       stimulating hormone (TSH) and chorionic gonadotropin (CG) are disclosed
       as exemplary mutant CKGFs. Mutant TSH heterodimers and hCH heterodimers
       possessed modified bioactivities, including superagonist activity.
       Accordingly, the present invention provides methods for using mutant
       CKGFs, CKGF analogs, fragments, and derivatives thereof for treating or
       preventing diseases. Pharmaceutical and diagnostic compositions, methods
       of using mutant TSH heterodimers and TSH analogs with utility for
       treatment and prevention of metabolic and reproductive diseases are also
       provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER:       2002:301743  USPATFULL
TITLE:                  Cystine knot growth factor mutants
INVENTOR(S):            Weintraub, Bruce D., Rockville, MD, UNITED STATES
                        Szkudlinski, Mariusz W., Potomac, MD, UNITED STATES

                             NUMBER        KIND    DATE
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PATENT INFORMATION:     US 2002169292      A1   20021114
APPLICATION INFO.:      US 2001-813398     A1   20010320  (9)
RELATED APPLN. INFO.:   Continuation of Ser. No. WO 1999-US5908, filed on 19
                        Mar 1999, UNKNOWN

                               NUMBER        DATE
                        ------------------ --------
PRIORITY INFORMATION:   WO 1998-US19772    19980922
DOCUMENT TYPE:          Utility
FILE SEGMENT:           APPLICATION
LEGAL REPRESENTATIVE:   Steven B. Kelber, PIPER, MARBURY RUDNICK & WOLFE,
                        L.L.P., 1200 Nineteenth Street N.W., Washington, DC,
                        20036-2412
NUMBER OF CLAIMS:       19
EXEMPLARY CLAIM:        1
NUMBER OF DRAWINGS:     20 Drawing Page(s)
LINE COUNT:             13856
CAS INDEXING IS AVAILABLE FOR THIS PATENT.


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L1          8757 S ALBUMIN FUSION PROTEIN?
L2           135 S BRAIN DERIVED NEUROTROPHIC FACTOR PROTEIN?
L3             0 S L1 AND L2
L4             1 S L2 AND FUSION

=> s l1 and stability
L5          2302 L1 AND STABILITY

=> s l5 and growth factor
L6            32 L5 AND GROWTH FACTOR

=>  e rosen, c/au
E1             7     ROSEN ZVI MICHAL/AU
E2             1     ROSEN ZWEIG JAMES/AU
E3             0 --> ROSEN, C/AU
E4             1     ROSENA BRUCE R/AU
E5             1     ROSENABUM S/AU
E6             1     ROSENACKER A F/AU
E7             1     ROSENACKER ARTHUR F/AU
E8             4     ROSENADA CEPERO R/AU
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E11           65     ROSENAK B D/AU
E12            9     ROSENAK BERNARD D/AU

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E1             1     HASELTINE WILLIAM G/AU
E2             1     HASELTINE WILLIAM GAGE/AU
E3             0 --> HASELTINE, W/AU
E4             1     HASELTLINE F P/AU
E5             5     HASELTON A/AU
E6             5     HASELTON AARON/AU
E7             1     HASELTON B J/AU
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E12           1      HASELTON CAROLE L/AU

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L6            32 S L5 AND GROWTH FACTOR
                 E ROSEN, C/AU
                 E HASELTINE, W/AU

=> s l6 and l2
L7             0 L6 AND L2
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=> s l1 and l2
L3            0 L1 AND L2

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L4            1 L2 AND FUSION

=> d l4 ti abs ibib tot

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                        L.L.P., 1200 Nineteenth Street N.W., Washington, DC,
                        20036-2412
NUMBER OF CLAIMS:       19
EXEMPLARY CLAIM:        1
NUMBER OF DRAWINGS:     20 Drawing Page(s)
LINE COUNT:             13856
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L6           32 S L5 AND GROWTH FACTOR
                E ROSEN, C/AU
                E HASELTINE, W/AU

=> s l6 and l2
L7            0 L6 AND L2

=> d l2 ti abs ibib 1-10

L2   ANSWER 1 OF 135     MEDLINE on STN
TI   Altered regulation of brain-derived
     neurotrophic factor protein in hippocampus
     following slice preparation.
AB   Brain-derived neurotrophic factor (BDNF) and its cognate receptor tyrosine
     kinase B (TrkB) play important roles in regulating survival, structure,
     and function of CNS neurons.  One method of studying the functions of
     these molecules has utilized in vitro hippocampal slice preparations.  An
     important caveat to using slices, however, is that slice preparation
     itself might alter the expression of BDNF, thereby confounding
     experimental results.  To address this concern, BDNF immunoreactivity was
     examined in rodent slices using two different methods of slice
     preparation.  Rapid and anatomically selective regulation of BDNF content
     followed slice preparation using both methodologies; however, different
     patterns of altered BDNF immunoreactivity were observed.  First, in
     cultured slices, BDNF content decreased in the dentate molecular layer and
     increased in the CA3 pyramidal cell layer and the mossy fiber pathway of
     the hippocampus after 30 min.  Furthermore, an initially "punctate"
     pattern of BDNF labeling observed in the mossy fiber pathway of control
     sections changed to homogenous labeling of the pathway in vitro.  In
     contrast to these findings, slices prepared as for acute slice physiology
     exhibited no change in BDNF content in the molecular layer and mossy fiber
     pathway 30 min after slicing, but exhibited significant increases in the
     dentate granule and CA3 pyramidal cell layers.  These findings demonstrate
     that BDNF protein content is altered following slice preparation, that
     different methods of slice preparation produce different patterns of BDNF
     regulation, and raise the possibility that BDNF release and TrkB
     activation may also be regulated.  These consequences of hippocampal slice
     preparation may confound analyses of exogenous or endogenous BDNF on
     hippocampal neuronal structure or function.
ACCESSION NUMBER:   2004306336     IN-PROCESS
DOCUMENT NUMBER:    PubMed ID: 15207321
TITLE:              Altered regulation of brain-derived
                    neurotrophic factor protein in
                    hippocampus following slice preparation.
AUTHOR:             Danzer S C; Pan E; Nef S; Parada L F; McNamara J O
CORPORATE SOURCE:   Department of Neurobiology, Duke University Medical Center,
                    401 Bryan Research Building, Durham, NC 27710, USA.
SOURCE:             Neuroscience, (2004) 126 (4) 859-69.
                    Journal code: 7605074. ISSN: 0306-4522.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
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LANGUAGE: English
FILE SEGMENT: IN-DATA-REVIEW; IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20040624
Last Updated on STN: 20040624

L2 ANSWER 2 OF 135 MEDLINE on STN
TI Prenatal cocaine exposure decreases **brain-derived neurotrophic factor proteins** in the rat brain.
AB The pregnant rats received daily sc injections of cocaine (30 mg/kg) or saline from the gestational day (GD) 7 to GD 20. At 1 week postnatal, all pups were killed and the hippocampus, cortex and striatum were dissected out. Levels of brain-derived neurotrophic factor (BDNF) under the basal condition and depolarization with high potassium (40 mM) were measured. The results showed that hippocampal BDNF levels under basal and depolarization conditions were all significantly lower in the pups prenatally exposed to cocaine than those exposed to saline. There were no significant differences in basal BDNF levels between the cocaine and saline groups in the cortex or striatum. However, the prenatally cocaine-treated pups showed significantly less BDNF release following high potassium depolarization than the saline-treated animals did in both these regions. The results support the suggestion that prenatal cocaine exposure decreases BDNF expression in the offspring.
ACCESSION NUMBER: 2004222297 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 15120602
TITLE: Prenatal cocaine exposure decreases **brain-derived neurotrophic factor proteins** in the rat brain.
AUTHOR: Yan Qing-Shan; Zheng Shi-Zhong; Yan Shu-E
CORPORATE SOURCE: Department of Biomedical and Therapeutic Sciences, University of Illinois College of Medicine, Peoria, IL 61656, USA.. QSY@UIC.EDU
SOURCE: Brain research, (2004 May 29) 1009 (1-2) 228-33. Journal code: 0045503. ISSN: 0006-8993.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20040505
Last Updated on STN: 20040616

L2 ANSWER 3 OF 135 MEDLINE on STN
TI Voluntary exercise protects against stress-induced decreases in **brain-derived neurotrophic factor protein** expression.
AB Exercise is increasingly recognized as an intervention that can reduce CNS dysfunctions such as cognitive decline, depression and stress. Previously we have demonstrated that brain-derived neurotrophic factor (BDNF) is increased in the hippocampus following exercise. In this study we tested the hypothesis that exercise can counteract a reduction in hippocampal BDNF protein caused by acute immobilization stress. Since BDNF expression is suppressed by corticosterone (CORT), circulating CORT levels were also monitored. In animals subjected to 2 h immobilization stress, CORT was elevated immediately following, and at 1 h after the cessation of stress, but remained unchanged from baseline up to 24 h post-stress. The stress protocol resulted in a reduction in BDNF protein at 5 and 10 h post-stress that returned to baseline at 24 h. To determine if exercise could prevent this stress-induced reduction in BDNF protein, animals were given voluntary access to running wheels for 3 weeks prior to the stress. Stressed animals, in the absence of exercise, again demonstrated an initial elevation in CORT (at 0 h) and a subsequent decrease in hippocampal BDNF at the 10 h time point. Exercising animals, both non-stressed and stressed, demonstrated circulating CORT and hippocampal BDNF protein levels that were significantly elevated above control values at both time points examined (0 and 10 h post-stress). Thus, the

persistently high CORT levels in exercised animals did not affect the induction of BDNF with exercise, and the effect of immobilization stress on BDNF protein was overcome. To examine the role of CORT in the stress-related regulation of BDNF protein, experiments were carried out in adrenalectomized (ADX) animals. BDNF protein was not downregulated as a result of immobilization stress in ADX animals, while there continued to be an exercise-induced upregulation of BDNF. This study demonstrates that CORT modulates stress-related alterations in BDNF protein. Further, exercise can override the negative effects of stress and high levels of CORT on BDNF protein. Voluntary physical activity may, therefore, represent a simple non-pharmacological tool for the maintenance of neurotrophin levels in the brain.

ACCESSION NUMBER: 2004134272 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15026138
TITLE: Voluntary exercise protects against stress-induced decreases in **brain-derived neurotrophic factor protein** expression.
AUTHOR: Adlard P A; Cotman C W
CORPORATE SOURCE: Institute for Brain Aging and Dementia, 1113 Gillespie N.R.F., University of California, Irvine, Irvine, CA 92697-4540, USA.. padlard@uci.edu
CONTRACT NUMBER: AG-13411-04 (NIA)
SOURCE: Neuroscience, (2004) 124 (4) 985-92.
Journal code: 7605074. ISSN: 0306-4522.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200406
ENTRY DATE: Entered STN: 20040318
Last Updated on STN: 20040609
Entered Medline: 20040608

L2 ANSWER 4 OF 135 MEDLINE on STN
TI Regulation of brain-derived neurotrophic factor (BDNF) expression following antibiotic treatment of experimental bacterial meningitis.
AB Although more and more new potent antibiotics have been used, mortality and neurologic deficits still occur frequently following bacterial meningitis in children. In this article, the expression of brain-derived neurotrophic factor messenger ribonucleic acid (RNA) and its production in the brains of rats were investigated during the course of experimental bacterial meningitis and after treatment with an antibiotic plus dexamethasone. In the brains of Streptococcus pneumoniae-inoculated rats, brain-derived neurotrophic factor (BDNF) messenger RNA was obviously up-regulated after inoculation for 24 hours ($P < .01$) and then declined but was still greater than that in the brains of control rats after inoculation for 5 days ($P < .05$). The expression of brain-derived neurotrophic factor in the brains of infected rats treated by antibiotic was dose dependent, down-regulated, and almost undetectable ($P < .01$) but up-regulated after treatment with an antibiotic plus dexamethasone ($P < .01$). However, the expression of brain-derived neurotrophic factor messenger RNA did not change in control rats treated with an antibiotic. **Brain-derived neurotrophic factor protein** showed similar changes, except it declined to normal levels 5 days after inoculation. Brain-derived neurotrophic factor messenger RNA and its production were observed in some infiltrating inflammatory cells in the brain of infected rats. The results of our studies support the hypothesis that brain-derived neurotrophic factor might play a neuroprotective role in brain damage during bacterial meningitis, and the expression of brain-derived neurotrophic factor messenger RNA and its production might be inhibited after treatment with antibiotics. The findings suggest that both eradicating the bacterial pathogen with antibiotics and adjuvant administering of brain-derived

neurotrophic factor might be more beneficial to prevent brain damage.

ACCESSION NUMBER: 2004036355 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14736076
TITLE: Regulation of brain-derived neurotrophic factor (BDNF) expression following antibiotic treatment of experimental bacterial meningitis.
AUTHOR: Li Ling; Shui Quan-Xiang; Zhao Zheng-Yan
CORPORATE SOURCE: Department of Neurology, Affiliated Children's Hospital, School of Medicine, Zhejiang University, Hangzhou, China.. hxyd_zjdx@sohu.com
SOURCE: Journal of child neurology, (2003 Dec) 18 (12) 828-34. Journal code: 8606714. ISSN: 0883-0738.
PUB. COUNTRY: Canada
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200403
ENTRY DATE: Entered STN: 20040123
Last Updated on STN: 20040330
Entered Medline: 20040329

L2 ANSWER 5 OF 135 MEDLINE on STN
TI Role of ubiquitin carboxy terminal hydrolase-L1 in neural cell apoptosis induced by ischemic retinal injury in vivo.
AB Ubiquitin is thought to be a stress protein that plays an important role in protecting cells under stress conditions; however, its precise role is unclear. Ubiquitin expression level is controlled by the balance of ubiquitinating and deubiquitinating enzymes. To investigate the function of deubiquitinating enzymes on ischemia-induced neural cell apoptosis in vivo, we analyzed gracile axonal dystrophy (gad) mice with an exon deletion for ubiquitin carboxy terminal hydrolase-L1 (UCH-L1), a neuron-specific deubiquitinating enzyme. In wild-type mouse retina, light stimuli and ischemic retinal injury induced strong ubiquitin expression in the inner retina, and its expression pattern was similar to that of UCH-L1. On the other hand, gad mice showed reduced ubiquitin induction after light stimuli and ischemia, whereas expression levels of antiapoptotic (Bcl-2 and XIAP) and prosurvival (**brain-derived neurotrophic factor**) **proteins** that are normally degraded by an ubiquitin-proteasome pathway were significantly higher. Consistently, ischemia-induced caspase activity and neural cell apoptosis were suppressed approximately 70% in gad mice. These results demonstrate that UCH-L1 is involved in ubiquitin expression after stress stimuli, but excessive ubiquitin induction following ischemic injury may rather lead to neural cell apoptosis in vivo.

ACCESSION NUMBER: 2003612189 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14695319
TITLE: Role of ubiquitin carboxy terminal hydrolase-L1 in neural cell apoptosis induced by ischemic retinal injury in vivo.
AUTHOR: Harada Takayuki; Harada Chikako; Wang Yu-Lai; Osaka Hitoshi; Amanai Kazuhito; Tanaka Kohichi; Takizawa Shuichi; Setsuie Rieko; Sakurai Mikako; Sato Yae; Noda Mami; Wada Keiji
CORPORATE SOURCE: Department of Degenerative Neurological Diseases, National Institute of Neuroscience, National Center of Neurology and Psychiatry, Kodaira, Tokyo, Japan.
SOURCE: American journal of pathology, (2004 Jan) 164 (1) 59-64. Journal code: 0370502. ISSN: 0002-9440.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200402
ENTRY DATE: Entered STN: 20031230
Last Updated on STN: 20040302

Entered Medline: 20040227

L2 ANSWER 6 OF 135 MEDLINE on STN
TI Effects of electroconvulsive seizures and antidepressant drugs on **brain-derived neurotrophic factor protein** in rat brain.
AB BACKGROUND: The antidepressant-like effects of brain-derived neurotrophic factor (BDNF) infusions in brain, and the upregulation of BDNF mRNA and its receptor in rats exposed to electroconvulsive seizure (ECS) and antidepressants, suggested a role for increased BDNF protein. METHODS: We measured BDNF protein levels with a two-site enzyme-linked immunosorbent assay (ELISA) in six brain regions of adult male rats that received daily ECS or daily injections of antidepressant drugs. RESULTS: The BDNF ELISA method was validated by the 50% loss of BDNF protein in the brains of +/- BDNF knockout mice, the 60%-100% recovery of spiked recombinant BDNF, and by the amounts and regional variations of BDNF measured in the six brain regions. Ten consecutive daily exposures to ECS increased BDNF protein in the parietal cortex (219%), entorhinal cortex (153%), hippocampus (132%), frontal cortex (94%), neostriatum (67%), and septum (29%). BDNF increased gradually in the hippocampus and frontal cortex, with a peak response by the fourth day of ECS. Increases peaked at 15 hours after the last ECS and lasted at least 3 days thereafter. Two weeks of daily injections with the monoamine (MAO)-A and -B inhibitor tranylcypromine (8-10 mg/kg, IP) increased BDNF by 15% in the frontal cortex, and 3 weeks treatment increased it by 18% in the frontal cortex and by 29% in the neostriatum. Tranylcypromine, fluoxetine, and desmethylimipramine did not elevate BDNF in the hippocampus. CONCLUSIONS: Elevations in BDNF protein in brain are consistent with the greater treatment efficacy of ECS and MAO inhibitors in drug-resistant major depressive disorder and may be predictive for the antidepressant action of the more highly efficacious interventions.
ACCESSION NUMBER: 2003449582 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14512210
TITLE: Effects of electroconvulsive seizures and antidepressant drugs on **brain-derived neurotrophic factor protein** in rat brain.
AUTHOR: Altar C Anthony; Whitehead Richard E; Chen Ruoyan; Wortwein Gitta; Madsen Torsten M
CORPORATE SOURCE: Global Neuroscience Research, Otsuka Maryland Research Institute, Inc., Rockville, Maryland, USA.
SOURCE: Biological psychiatry, (2003 Oct 1) 54 (7) 703-9. Journal code: 0213264. ISSN: 0006-3223.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200310
ENTRY DATE: Entered STN: 20030928
Last Updated on STN: 20031024
Entered Medline: 20031023

L2 ANSWER 7 OF 135 MEDLINE on STN
TI Anterograde delivery of brain-derived neurotrophic factor to striatum via nigral transduction of recombinant adeno-associated virus increases neuronal death but promotes neurogenic response following stroke.
AB To explore the role of brain-derived neurotrophic factor for survival and generation of striatal neurons after stroke, recombinant adeno-associated viral vectors carrying brain-derived neurotrophic factor or green fluorescent protein genes were injected into right rat substantia nigra 4-5 weeks prior to 30 min ipsilateral of middle cerebral artery occlusion. The brain-derived neurotrophic factor-recombinant adeno-associated viral transduction markedly increased the production of **brain-derived neurotrophic factor protein** by nigral cells. Brain-derived neurotrophic factor was transported

anterogradely to the striatum and released in biologically active form, as revealed by the hypertrophic response of striatal neuropeptide Y-positive interneurons. Animals transduced with brain-derived neurotrophic factor-recombinant adeno-associated virus also exhibited abnormalities in body posture and movements, including tilted body to the right, choreiform movements of left forelimb and head, and spontaneous, so-called 'barrel' rotation along their long axis. The continuous delivery of brain-derived neurotrophic factor had no effect on the survival of striatal projection neurons after stroke, but exaggerated the loss of cholinergic, and parvalbumin- and neuropeptide Y-positive, gamma-aminobutyric acid-ergic interneurons. The high brain-derived neurotrophic factor levels in the animals subjected to stroke also gave rise to an increased number of striatal cells expressing doublecortin, a marker for migrating neuroblasts, and cells double-labelled with the mitotic marker, 5-bromo-2'-deoxyuridine-5'monophosphate, and early neuronal (Hu) or striatal neuronal (Meis2) markers. Our findings indicate that long-term anterograde delivery of high levels of brain-derived neurotrophic factor increases the vulnerability of striatal interneurons to stroke-induced damage. Concomitantly, brain-derived neurotrophic factor potentiates the stroke-induced neurogenic response, at least at early stages.

ACCESSION NUMBER: 2003347254 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12823474
TITLE: Anterograde delivery of brain-derived neurotrophic factor to striatum via nigral transduction of recombinant adeno-associated virus increases neuronal death but promotes neurogenic response following stroke.
AUTHOR: Gustafsson Elin; Andsberg Gunnar; Darsalia Vladimer; Mohapel Paul; Mandel Ronald J; Kirik Deniz; Lindvall Olle; Kokaia Zaal
CORPORATE SOURCE: Section of Restorative Neurology, Wallenberg Neuroscience Center, University of Lund, BMC A-11 SE-221 84 Lund, Sweden.
SOURCE: European journal of neuroscience, (2003 Jun) 17 (12) 2667-78.
Journal code: 8918110. ISSN: 0953-816X.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200309
ENTRY DATE: Entered STN: 20030726
Last Updated on STN: 20030925
Entered Medline: 20030924

L2 ANSWER 8 OF 135 MEDLINE on STN
TI Single eight-hour shift of light-dark cycle increases **brain-derived neurotrophic factor protein** levels in the rat hippocampus.
AB We previously reported that an eight hour phase advance in the light-dark (LD) cycle increases sleep in rats. Brain-derived neurotrophic factor (BDNF) is suggested to be one of the sleep and circadian regulating factors. We have therefore observed the responses of BDNF protein in the hippocampus, cerebellum and brainstem under conditions of LD change. BDNF protein was quantitatively measured using an ELISA kit. Under an 8-h LD phase advance, the levels of hippocampal BDNF were significantly increased on the day of the phase change, while the levels in the cerebellum and brainstem remained constant. Plasma corticosterone levels were not largely affected. Thus, a single LD shift acutely affects hippocampal BDNF metabolism with no large stress response.

ACCESSION NUMBER: 2003206870 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12726886
TITLE: Single eight-hour shift of light-dark cycle increases **brain-derived neurotrophic factor protein** levels in the rat

hippocampus.
AUTHOR: Sei Hiroyoshi; Fujihara Hiroaki; Ueta Yoichi; Morita Kyoji; Kitahama Kunio; Morita Yusuke
CORPORATE SOURCE: Department of Integrative Physiology, School of Medicine, The University of Tokushima, Tokushima 770-8503, Japan.. sei@basic.med.tokushima-u.ac.jp
SOURCE: Life sciences, (2003 May 23) 73 (1) 53-9.
Journal code: 0375521. ISSN: 0024-3205.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200305
ENTRY DATE: Entered STN: 20030503
Last Updated on STN: 20030530
Entered Medline: 20030529

L2 ANSWER 9 OF 135 MEDLINE on STN
TI Activity-dependent change in the protein level of brain-derived neurotrophic factor but no change in other neurotrophins in the visual cortex of young and adult ferrets.
AB Neurotrophins are suggested to play a role in activity-dependent plasticity of visual cortex during the critical period of postnatal development. Thus, the concentration of neurotrophins in the cortex is expected to change with development and/or with alteration in neuronal activities. To test this, we measured protein levels of nerve growth factor, brain-derived neurotrophic factor, neurotrophin-3 and neurotrophin-4/5 in visual cortex of young (postnatal day 38-46, at the peak of the critical period) and adult ferrets with two-site enzyme-immunoassay systems. Measurements were carried out also in somatosensory cortex, hippocampus and cerebellum as control. With development the level of brain-derived neurotrophic factor did not significantly change, while those of the other neurotrophins changed in the visual cortex. A blockade of visual inputs for 24 h by an injection of tetrodotoxin into both eyes significantly decreased **brain-derived neurotrophic factor protein** level in the visual cortex, but not in the other regions in both young and adult ferrets. On the other hand, no significant decrease was seen in the protein level of the other neurotrophins in the visual cortex of young and adult ferrets. A monocular injection of tetrodotoxin in young ferrets resulted in the reduction of brain-derived neurotrophic factor by approximately half that by binocular injection. The degree of the decrease in the contralateral cortex to the injected eye was significantly larger than that in the ipsilateral cortex, reflecting that the contralateral eye is dominantly represented in the cortex in ferrets. Blockade of cortical neuronal activities by a GABA(A) receptor agonist led to a remarkable reduction of **brain-derived neurotrophic factor protein** in the visual cortex. These results suggest that the level of **brain-derived neurotrophic factor protein** in visual cortex is regulated by activities of cortical neurons.
ACCESSION NUMBER: 2003103423 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12614676
TITLE: Activity-dependent change in the protein level of brain-derived neurotrophic factor but no change in other neurotrophins in the visual cortex of young and adult ferrets.
AUTHOR: Ichisaka S; Katoh-Semba R; Hata Y; Ohshima M; Kameyama K; Tsumoto T
CORPORATE SOURCE: Division of Neurophysiology, Osaka University Graduate School of Medicine, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan.
SOURCE: Neuroscience, (2003) 117 (2) 361-71.
Journal code: 7605074. ISSN: 0306-4522.

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200305
ENTRY DATE: Entered STN: 20030305
Last Updated on STN: 20030522
Entered Medline: 20030521

L2 ANSWER 10 OF 135 MEDLINE on STN
TI Time-dependent increases in **brain-derived neurotrophic factor protein** levels within the mesolimbic dopamine system after withdrawal from cocaine: implications for incubation of cocaine craving.
AB Using a rat model of drug craving, we found that the responsiveness to cocaine cues progressively increases or incubates over the first 60 d of cocaine withdrawal. Here we studied whether alterations in brain-derived neurotrophic factor (BDNF) protein levels within the mesolimbic dopamine system are associated with this incubation phenomenon. BDNF is involved in synaptic plasticity and was found to enhance responding for cues associated with natural rewards. Rats were trained to press a lever to receive intravenous cocaine or oral sucrose for 6 hr/d for 10 d; each earned reward was paired with a tone-light cue. Resumption of lever-pressing behavior was then assessed on days 1, 30, or 90 of reward withdrawal. First, resistance to extinction was assessed during 6 hr in which lever presses were not reinforced and the cue was absent. Second, cue-induced reinstatement was assessed after extinction during 1 hr in which responding led to cue presentations. Other rats were killed without testing on days 1, 30, and 90 of reward withdrawal, and BDNF and nerve growth factor (NGF) protein levels were measured in the ventral tegmental area (VTA), accumbens, and amygdala. Lever pressing during extinction and cue-induced reinstatement tests of cocaine craving progressively increased after cocaine withdrawal. Time-dependent changes also were observed during the tests for sucrose craving, with maximal responding on day 30. BDNF, but not NGF, levels in the VTA, accumbens, and amygdala progressively increased after cocaine, but not sucrose, withdrawal. Time-dependent increases in BDNF levels may lead to synaptic modifications that underlie enhanced responsiveness to cocaine cues after prolonged withdrawal periods.
ACCESSION NUMBER: 2003064020 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12574402
TITLE: Time-dependent increases in **brain-derived neurotrophic factor protein** levels within the mesolimbic dopamine system after withdrawal from cocaine: implications for incubation of cocaine craving.
AUTHOR: Grimm Jeffrey W; Lu Lin; Hayashi Teruo; Hope Bruce T; Su Tsung-Ping; Shaham Yavin
CORPORATE SOURCE: Behavioral Neuroscience Branch, Intramural Research Program/National Institute on Drug Abuse/National Institutes of Health/Department of Health and Human Services, Baltimore, Maryland 21224, USA.
SOURCE: Journal of neuroscience : official journal of the Society for Neuroscience, (2003 Feb 1) 23 (3) 742-7.
Journal code: 8102140. ISSN: 1529-2401.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20030208
Last Updated on STN: 20030222
Entered Medline: 20030221

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     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, BIOSIS, HCAPLUS'
     ENTERED AT 18:02:51 ON 30 JUN 2004
L1          8757 S ALBUMIN FUSION PROTEIN?
L2           135 S BRAIN DERIVED NEUROTROPHIC FACTOR PROTEIN?
L3             0 S L1 AND L2
L4             1 S L2 AND FUSION
L5          2302 S L1 AND STABILITY
L6            32 S L5 AND GROWTH FACTOR
                 E ROSEN, C/AU
                 E HASELTINE, W/AU
L7             0 S L6 AND L2

=> s l2 and stability
L8             1 L2 AND STABILITY

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'OT' IS NOT A VALID FORMAT FOR FILE 'USPATFULL'

The following are valid formats:

The default display format is STD.

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ALL -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
             RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
             DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL,
             INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
             EXF, ARTU
ALLG ------- ALL plus PAGE.DRAW
BIB -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD, RLI,
             PRAI, DT, FS, EXNAM, LREP, CLMN, ECL, DRWN, LN.CNT
BIB.EX ----- BIB for original and latest publication
BIBG ------- BIB plus PAGE.DRAW
BROWSE ----- See "HELP BROWSE" or "HELP DISPLAY BROWSE".  BROWSE must
             entered on the same line as DISPLAY, e.g., D BROWSE.
CAS -------- OS, CC, SX, ST, IT
CBIB ------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PRAI, DT, FS
DALL ------- ALL, delimited for post-processing
FP --------- PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI, RLI,
             PRAI, IC, ICM, ICS, INCL, INCLM, INCLS, NCL,
             NCLM, NCLS, EXF, REP, REN, ARTU, EXNAM, LREP,
             CLMN, DRWN, AB
FP.EX ------ FP for original and latest publication
FPALL ------ PI, TI, IN, INA, PA, PAA, PAT, PETRM, DCD, AI,
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             PARN, SUMM, DRWD, DETD, CLM
FPBIB ------ PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI,
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FHITSTR ---- HIT RN, its text modification, its CA index name, and
             its structure diagram
FPG -------- FP plus PAGE.DRAW
GI --------- PN and page image numbers
HIT -------- All fields containing hit terms
HITRN ------ HIT RN and its text modification
HITSTR ----- HIT RN, its text modification, its CA index name, and
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IABS ------- ABS, indented with text labels
IALL ------- ALL, indented with text labels
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IALLG ------ IALL plus PAGE.DRAW
IBIB ------- BIB, indented with text labels
IBIB.EX ---- IBIB for original and latest publication
IBIBG ------ IBIB plus PAGE.DRAW
IMAX ------- MAX, indented with text labels
IMAX.EX ---- IMAX for original and latest publication
IND -------- INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
             EXF, ARTU, OS, CC, SX, ST, IT
ISTD ------- STD, indented with text labels
KWIC ------- All hit terms plus 20 words on either side
MAX -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
             RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
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             INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
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MAX.EX ----- MAX for original and latest publication
OCC -------- List of display fields containing hit terms
SBIB ------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
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SCAN ------- AN, TI, NCL, NCLM, NCLS, IC, ICM, ICS (random display
             without answer number.  SCAN must be entered on the
             same line as DISPLAY, e.g., D SCAN)
STD -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
             DT, FS, LN.CNT, INCL, INCLM, INCLS, NCL, NCLM, NCLS,
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STD.EX ----- STD for original and latest publication
TRIAL ------ AN, TI, INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC,
             ICM, ICS

ENTER DISPLAY FORMAT (STD):d his
'D' IS NOT A VALID FORMAT FOR FILE 'USPATFULL'
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The following are valid formats:

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ABS -------- AB
ALL -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
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             EXF, ARTU
ALLG ------- ALL plus PAGE.DRAW
BIB -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD, RLI,
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BROWSE ----- See "HELP BROWSE" or "HELP DISPLAY BROWSE".  BROWSE must
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CBIB ------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PRAI, DT, FS
DALL ------- ALL, delimited for post-processing
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FP.EX ------ FP for original and latest publication
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             PARN, SUMM, DRWD, DETD, CLM
FPBIB ------ PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI,
             RLI, PRAI, REP, REN, EXNAM, LREP, CLM, CLMN, DRWN
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GI --------- PN and page image numbers
HIT -------- All fields containing hit terms
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HITSTR ----- HIT RN, its text modification, its CA index name, and
             its structure diagram
IABS ------- ABS, indented with text labels
IALL ------- ALL, indented with text labels
IALLG ------ IALL plus PAGE.DRAW
IBIB ------- BIB, indented with text labels
IBIB.EX ---- IBIB for original and latest publication
IBIBG ------ IBIB plus PAGE.DRAW
IMAX ------- MAX, indented with text labels
IMAX.EX ---- IMAX for original and latest publication
IND -------- INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
             EXF, ARTU, OS, CC, SX, ST, IT
ISTD ------- STD, indented with text labels
KWIC ------- All hit terms plus 20 words on either side
MAX -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
             RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
             DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL,
             INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
             EXF, ARTU OS, CC, SX, ST, IT
MAX.EX ----- MAX for original and latest publication
OCC -------- List of display fields containing hit terms
SBIB ------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
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SCAN ------- AN, TI, NCL, NCLM, NCLS, IC, ICM, ICS (random display
             without answer number.  SCAN must be entered on the
             same line as DISPLAY, e.g., D SCAN)
STD -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
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STD.EX ----- STD for original and latest publication
TRIAL ------ AN, TI, INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC,
             ICM, ICS

ENTER DISPLAY FORMAT (STD):end

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     (FILE 'HOME' ENTERED AT 18:01:32 ON 30 JUN 2004)

     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, BIOSIS, HCAPLUS'
     ENTERED AT 18:02:51 ON 30 JUN 2004
L1          8757 S ALBUMIN FUSION PROTEIN?
L2           135 S BRAIN DERIVED NEUROTROPHIC FACTOR PROTEIN?
L3             0 S L1 AND L2
L4             1 S L2 AND FUSION
L5          2302 S L1 AND STABILITY
L6            32 S L5 AND GROWTH FACTOR
                 E ROSEN, C/AU
                 E HASELTINE, W/AU
L7             0 S L6 AND L2
L8             1 S L2 AND STABILITY

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L8   ANSWER 1 OF 1  USPATFULL on STN
TI     Cystine knot growth factor mutants
AB     Compositions and methods based on mutant Cystine Knot Growth Factors
       (CKGFs) comprising amino acid substitutions relative to the wild type
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hormone/growth factor. Mutated glycoprotein hormones, including thyroid stimulating hormone (TSH) and chorionic gonadotropin (CG) are disclosed as exemplary mutant CKGFs. Mutant TSH heterodimers and hCH heterodimers possessed modified bioactivities, including superagonist activity. Accordingly, the present invention provides methods for using mutant CKGFs, CKGF analogs, fragments, and derivatives thereof for treating or preventing diseases. Pharmaceutical and diagnostic compositions, methods of using mutant TSH heterodimers and TSH analogs with utility for treatment and prevention of metabolic and reproductive diseases are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2002:301743 USPATFULL
TITLE:	Cystine knot growth factor mutants
INVENTOR(S):	Weintraub, Bruce D., Rockville, MD, UNITED STATES Szkudlinski, Mariusz W., Potomac, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002169292	A1	20021114
APPLICATION INFO.:	US 2001-813398	A1	20010320 (9)
RELATED APPLN. INFO.:	Continuation of Ser. No. WO 1999-US5908, filed on 19 Mar 1999, UNKNOWN		

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1998-US19772	19980922

DOCUMENT TYPE:	Utility
FILE SEGMENT:	APPLICATION
LEGAL REPRESENTATIVE:	Steven B. Kelber, PIPER, MARBURY RUDNICK & WOLFE, L.L.P., 1200 Nineteenth Street N.W., Washington, DC, 20036-2412
NUMBER OF CLAIMS:	19
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	20 Drawing Page(s)
LINE COUNT:	13856

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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     ENTERED AT 18:02:51 ON 30 JUN 2004
L1          8757 S ALBUMIN FUSION PROTEIN?
L2           135 S BRAIN DERIVED NEUROTROPHIC FACTOR PROTEIN?
L3             0 S L1 AND L2
L4             1 S L2 AND FUSION
L5          2302 S L1 AND STABILITY
L6            32 S L5 AND GROWTH FACTOR
                 E ROSEN, C/AU
                 E HASELTINE, W/AU
L7             0 S L6 AND L2
L8             1 S L2 AND STABILITY

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L9             3 L2 AND STABL?

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The following are valid formats:
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The default display format is STD.

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             RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
             DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL,
             INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
             EXF, ARTU
ALLG ------- ALL plus PAGE.DRAW
BIB -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD, RLI,
             PRAI, DT, FS, EXNAM, LREP, CLMN, ECL, DRWN, LN.CNT
BIB.EX ----- BIB for original and latest publication
BIBG ------- BIB plus PAGE.DRAW
BROWSE ----- See "HELP BROWSE" or "HELP DISPLAY BROWSE".  BROWSE must
             entered on the same line as DISPLAY, e.g., D BROWSE.
CAS -------- OS, CC, SX, ST, IT
CBIB ------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PRAI, DT, FS
DALL ------- ALL, delimited for post-processing
FP --------- PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI, RLI,
             PRAI, IC, ICM, ICS, INCL, INCLM, INCLS, NCL,
             NCLM, NCLS, EXF, REP, REN, ARTU, EXNAM, LREP,
             CLMN, DRWN, AB
FP.EX ------ FP for original and latest publication
FPALL ------ PI, TI, IN, INA, PA, PAA, PAT, PETRM, DCD, AI,
             RLI, PRAI, IC, ICM, ICS, INCL, INCLM, INCLS, NCL, NCLM,
             NCLS, EXF, REP, REN,ARTU, EXNAM, LREP, CLMN, DRWN, AB,
             PARN, SUMM, DRWD, DETD, CLM
FPBIB ------ PI, TI, IN, INA, PA, PAA, PAT, PTERM, DCD, AI,
             RLI, PRAI, REP, REN, EXNAM, LREP, CLM, CLMN, DRWN
FHITSTR ---- HIT RN, its text modification, its CA index name, and
             its structure diagram
FPG -------- FP plus PAGE.DRAW
GI --------- PN and page image numbers
HIT -------- All fields containing hit terms
HITRN ------ HIT RN and its text modification
HITSTR ----- HIT RN, its text modification, its CA index name, and
             its structure diagram
IABS ------- ABS, indented with text labels
IALL ------- ALL, indented with text labels
IALLG ------ IALL plus PAGE.DRAW
IBIB ------- BIB, indented with text labels
IBIB.EX ---- IBIB for original and latest publication
IBIBG ------ IBIB plus PAGE.DRAW
IMAX ------- MAX, indented with text labels
IMAX.EX ---- IMAX for original and latest publication
IND -------- INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
             EXF, ARTU, OS, CC, SX, ST, IT
ISTD ------- STD, indented with text labels
KWIC ------- All hit terms plus 20 words on either side
MAX -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, PTERM, DCD,
             RLI, PRAI, DT, FS, REP, REN, EXNAM, LREP, CLMN, ECL,
             DRWN, AB, GOVI, PARN, SUMM, DRWD, DETD, CLM, INCL,
             INCLM, INCLS, NCL, NCLM, NCLS, IC, ICM, ICS,
             EXF, ARTU OS, CC, SX, ST, IT
MAX.EX ----- MAX for original and latest publication
OCC -------- List of display fields containing hit terms
SBIB ------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
             DT, FS, LN.CNT
SCAN ------- AN, TI, NCL, NCLM, NCLS, IC, ICM, ICS (random display
             without answer number.  SCAN must be entered on the
             same line as DISPLAY, e.g., D SCAN)
STD -------- AN, TI, IN, INA, PA, PAA, PAT, PI, AI, RLI, PRAI,
             DT, FS, LN.CNT, INCL, INCLM, INCLS, NCL, NCLM, NCLS,
             IC, ICM, ICS, EXF (STD is the default)
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STD.EX ----- STD for original and latest publication
TRIAL ------ AN, TI, INCL, INCLM, INCLS, NCL, NCLM, NCLS, IC,
             ICM, ICS

ENTER DISPLAY FORMAT (STD):end

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     (FILE 'HOME' ENTERED AT 18:01:32 ON 30 JUN 2004)

     FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, WPIDS, FSTA, BIOSIS, HCAPLUS'
     ENTERED AT 18:02:51 ON 30 JUN 2004
L1          8757 S ALBUMIN FUSION PROTEIN?
L2           135 S BRAIN DERIVED NEUROTROPHIC FACTOR PROTEIN?
L3             0 S L1 AND L2
L4             1 S L2 AND FUSION
L5          2302 S L1 AND STABILITY
L6            32 S L5 AND GROWTH FACTOR
                 E ROSEN, C/AU
                 E HASELTINE, W/AU
L7             0 S L6 AND L2
L8             1 S L2 AND STABILITY
L9             3 S L2 AND STABL?

=> d l9 ti abs ibib tot

L9   ANSWER 1 OF 3  USPATFULL on STN
TI     Cystine knot growth factor mutants
AB     Compositions and methods based on mutant Cystine Knot Growth Factors
       (CKGFs) comprising amino acid substitutions relative to the wild type
       hormone/growth factor. Mutated glycoprotein hormones, including thyroid
       stimulating hormone (TSH) and chorionic gonadotropin (CG) are disclosed
       as exemplary mutant CKGFs. Mutant TSH heterodimers and hCH heterodimers
       possessed modified bioactivities, including superagonist activity.
       Accordingly, the present invention provides methods for using mutant
       CKGFs, CKGF analogs, fragments, and derivatives thereof for treating or
       preventing diseases. Pharmaceutical and diagnostic compositions, methods
       of using mutant TSH heterodimers and TSH analogs with utility for
       treatment and prevention of metabolic and reproductive diseases are also
       provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER:        2002:301743  USPATFULL
TITLE:                   Cystine knot growth factor mutants
INVENTOR(S):             Weintraub, Bruce D., Rockville, MD, UNITED STATES
                         Szkudlinski, Mariusz W., Potomac, MD, UNITED STATES

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 2002169292      A1   20021114
APPLICATION INFO.:       US 2001-813398     A1   20010320  (9)
RELATED APPLN. INFO.:    Continuation of Ser. No. WO 1999-US5908, filed on 19
                         Mar 1999, UNKNOWN

                                NUMBER         DATE
                         ------------------ --------
PRIORITY INFORMATION:    WO 1998-US19772    19980922
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    Steven B. Kelber, PIPER, MARBURY RUDNICK & WOLFE,
                         L.L.P., 1200 Nineteenth Street N.W., Washington, DC,
                         20036-2412
NUMBER OF CLAIMS:        19
EXEMPLARY CLAIM:         1
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NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 13856
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 2 OF 3 USPATFULL on STN
TI Brain derived neurotrophic factor
AB The present invention relates to nucleic acid sequences encoding brain derived neurotrophic factor (BDNF), as well as BDNF protein produced in quantity using these nucleic acid sequences, as well as fragments and derivatives thereof. In addition, the invention relates to pharmacologic compositions and therapeutic uses of BDNF, having provided, for the first time, the means to generate sufficient quantities of substantially pure BDNF for clinical use. In a specific embodiment, BDNF may be used to promote the survival of substantia nigra dopaminergic neurons and basal forebrain cholinergic neurons, thereby providing a method for treating, respectively, Parkinson's disease and Alzheimer's disease. The invention also relates to antibodies directed toward BDNF or fragments thereof, having provided a method for generating sufficient immunogen. Further, by permitting a comparison of the nucleic acid sequences of BDNF and NGF, the present invention provides for the identification of homologous regions of nucleic acid sequence between BDNF and NGF, thereby defining a BDNF/NGF gene family; the invention provides a method for identifying and isolating additional members of this gene family.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 95:69347 USPATFULL
TITLE: Brain derived neurotrophic factor
INVENTOR(S): Barde, Yves-Alain, Munich, Germany, Federal Republic of
Leibrock, Joachim, Gauting, Germany, Federal Republic of
Lottspeich, Friedrich, Neuried, Germany, Federal Republic of
Edgar, David, Liverpool, England
Yancopoulos, George, New York, NY, United States
Thoenen, Hans, Munich, Germany, Federal Republic of
PATENT ASSIGNEE(S): Max-Planck-Gesellschaft zur Foderund der Wissenschaften e.V., Martinsfried, Germany, Federal Republic of (non-U.S. corporation)
Regeneron Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5438121		19950801	
APPLICATION INFO.:	US 1991-691612		19910425	(7)

Considered

DISCLAIMER DATE: 20100720
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1990-570657, filed on 20 Aug 1990, now patented, Pat. No. US 5229500 which is a continuation-in-part of Ser. No. US 1989-400591, filed on 30 Aug 1989, now patented, Pat. No. US 5180820
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Hill, Jr., Robert J.
ASSISTANT EXAMINER: Wang, Gian P.
LEGAL REPRESENTATIVE: Pennie & Edmonds
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 68 Drawing Figure(s); 52 Drawing Page(s)
LINE COUNT: 5042
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 3 OF 3 USPATFULL on STN
TI Brain derived neurotrophic factor
AB The present invention relates to nucleic acid sequences encoding brain

derived neurotrophic factor (BDNF), as well as BDNF protein produced in quantity using these nucleic acid sequences, as well as fragments and derivatives thereof. In addition, the invention relates to pharmacologic compositions and therapeutic uses of BDNF, having provided, for the first time, the means to generate sufficient quantities of substantially pure BDNF for clinical use. In a specific embodiment, BDNF may be used to promote the survival of substantia nigra dopaminergic neurons and basal forebrain cholinergic neurons, thereby providing a method for treating, respectively, Parkinson's disease and Alzheimer's disease. The invention also relates to antibodies directed toward BDNF or fragments thereof, having provided a method for generating sufficient immunogen. Further, by permitting a comparison of the nucleic acid sequences of BDNF and NGF, the present invention provides for the identification of homologous regions of nucleic acid sequence between BDNF and NGF, thereby defining a BDNF/NGF gene family; the invention provides a method for identifying and isolating additional members of this gene family.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	93:59268 USPATFULL
TITLE:	Brain derived neurotrophic factor
INVENTOR(S):	Barde, Yves-Alain, Graefelfing, Germany, Federal Republic of Leibrock, Joachim, Pfungstadt, Germany, Federal Republic of Lottspeich, Friedrich, Neuried, Germany, Federal Republic of Edgar, David, Liverpool, England Yancopoulos, George, Briarcliff Manor, NY, United States Thoenen, Hans, Munich, Germany, Federal Republic of
PATENT ASSIGNEE(S):	Regeneron Pharmaceuticals, Inc., Tarrytown, NY, United States (U.S. corporation) Max Planck Gesellschaft, Martinsried, Germany, Federal Republic of (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5229500		19930720
APPLICATION INFO.:	US 1990-570657		19900820 (7)

considered HK

RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1989-400591, filed on 30 Aug 1989, now patented, Pat. No. US 5180820
DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Hill, Jr., Robert J.
ASSISTANT EXAMINER:	Wang, Gian P.
LEGAL REPRESENTATIVE:	Pennie & Edmonds
NUMBER OF CLAIMS:	9
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	66 Drawing Figure(s); 51 Drawing Page(s)
LINE COUNT:	4439

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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 file medline
COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                          ENTRY    SESSION
FULL ESTIMATED COST                                       27.70      28.12

FILE 'MEDLINE' ENTERED AT 18:09:33 ON 30 JUN 2004

 FILE LAST UPDATED: 29 JUN 2004 (20040629/UP).  FILE COVERS 1951 TO DATE.

 On February 29, 2004, the 2004 MeSH terms were loaded.  See HELP RLOAD
 for details. OLDMEDLINE now back to 1951.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2004 vocabulary.  See http://www.nlm.nih.gov/mesh/ and
 http://www.nlm.nih.gov/pubs/techbull/nd03/nd03_mesh.html for a
 description of changes.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> s brain derived neurotrophic factor protein+nt/CT
'BRAIN DERIVED NEUROTROPHIC FACTOR PROTEIN' NOT IN RELATIONSHIP FILE
RELATIONSHIP CODE 'NT' IGNORED
L10             0 BRAIN DERIVED NEUROTROPHIC FACTOR PROTEIN+NT/CT  (1 TERM)

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Refine Search

Search Results -

Terms	Documents
L17 and BDNF	0

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L18

Refine Search

Recall Text Clear Interrupt

Search History

DATE: Wednesday, June 30, 2004 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L18	L17 and BDNF	0	L18
L17	L16 and l15	11	L17
L16	l13 and l3	368	L16
L15	L14 and l3	34	L15
L14	haseltine.in.	329	L14
L13	rosen.in.	2229	L13
L12	L11 and l1	1	L12
L11	L10 and l3	139307	L11
L10	brain derived neurotrophic factor protein adj2 albumin	931351	L10
L9	BDNF adj2 albumin	0	L9
L8	"B5 peptide" and l1	1	L8
L7	l1 and fusion	0	L7
L6	l1 and albumin	1	L6
L5	l3 and l2	1	L5

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L4	l1 and L3	1	L4
L3	albumin fusion protein	204485	L3
L2	5229500.pn.	1	L2
L1	5438121.pn.	1	L1

END OF SEARCH HISTORY

First Hit Fwd Refs

End of Result Set

Generate Collection Print

L8: Entry 1 of 1 File: USPT Aug 1, 1995

DOCUMENT-IDENTIFIER: US 5438121 A
TITLE: Brain derived neurotrophic factor

Drawing Description Text (8):
FIG. 6A. Results of ELISA determination of binding of antisera to B5 peptide, using serial dilutions of antisera.

Detailed Description Text (164):
The B5 peptide was coupled to bovine serum albumin (BSA) using bis-diazo benzidine (BDB). Fresh BDB was prepared by dissolving 46 mg benzidine-HCl (p-diaminodiphenyl-HCl, obtained from Sigma) in 9.0 ml of 0.2 N HCl. 35 mg NaNO.sup.2 was dissolved in 1.0 ml H.sub.2 O and added to the benzidine solution, and stirred for 1 hour at 4.degree. C. 21 mg of BSA was dissolved in 3.0 ml of 0.16M borate, 0.13 M NaCl, pH 9.0. Approximately 15 mg of B5 peptide was dissolved in 1.5 ml borate-NaCl buffer, pH 0.0. The peptide solution was added to the BSA solution, and placed in ice. 1.0 ml of BDB was added to the BSA-peptide solution, and the reaction mixture was incubated with stirring for 2 hours at 4.degree. C.; the pH was monitored and maintained in the range of 9.0 by the addition of small amounts of 0.5M NAOH, as required. The reaction was terminated by addition of 0.2 ml of 1% phenol-buffered solution. Excess reagents were removed by dialysis against phosphate buffered saline (PBS).

Detailed Description Text (170):
In all cases the first immunization used 1 mg of immunogen (100 .mu.g B5/500 .mu.g nitrocellulose for rabbits 5 and 6) in 0.5 ml PBS plus 0.5 ml ml complete Freund's adjuvant. This mixture was injected subcutaneously into multiple sites on the back. The second immunization was carried out three weeks later, and was identical to the first except that incomplete Freund's adjuvant was used in place of complete Freund's adjuvant. Subsequent boosts occurred at intervals of 4-6 weeks. Rabbits were bled 1 week after immunization, and the antisera routinely checked for binding to the pure B5 peptide by enzyme-linked immunosorbent assay (ELISA).

Detailed Description Text (172):
100 .mu.g of antigen (B5 peptide) in H.sub.2 O was added to wells on a microtiter plate and allowed to dry overnight, then washed briefly with H.sub.2 O and blocked with 100 .mu.g 1% gelatin for 30 minutes at room temperature. Wells were washed three times with distilled water, and then 100 .mu.g of antisera was added and allowed to incubate at 4.degree. C. overnight. Wells were then washed three times in PBS/0.05% triton X-100, after which 100 .mu.g peroxidase labeled anti-rabbit immunoassay (1/1000 dilution) was added to wells and incubated at room temperature for three hours. Wells were washed twice, and 100 .mu.g ABTS solution (10 mg ABTS (Sigma) dissolved in 10 ml 0.1M NaCitrate pH 4.0 plus 10 .mu.g H.sub.2 O.sub.2) was added and incubated for about 5 minutes, until color developed. The reaction was stopped by the addition of 10 .mu.g 1% NaN.sub. 3. Samples were diluted 1:5 with H.sub.2 O and optical density was measured at 415 nm.

Hit List

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Search Results - Record(s) 1 through 10 of 11 returned.

☐ 1. Document ID: US 6620619 B2

L17: Entry 1 of 11 File: USPT Sep 16, 2003

US-PAT-NO: 6620619
DOCUMENT-IDENTIFIER: US 6620619 B2

TITLE: Human DNA mismatch repair protein

DATE-ISSUED: September 16, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Haseltine; William A.	Washington	DC		
Ruben; Steven	Olney	MD		
Wei; Ying-Fei	Darnestown	MD		
Adams; Mark D.	North Potomac	MD		
Fleischmann; Robert D.	Washington	DC		
Fraser; Claire M.	Queenstown	MD		
Rosen; Craig A.	Laytonsville	MD		
Fuldner; Rebecca A.	Barnesville	MD		
Kirkness; Ewen F.	Washington	DC		

US-CL-CURRENT: 536/23.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw De

☐ 2. Document ID: US 6610477 B1

L17: Entry 2 of 11 File: USPT Aug 26, 2003

US-PAT-NO: 6610477
DOCUMENT-IDENTIFIER: US 6610477 B1

TITLE: Human DNA mismatch repair proteins

DATE-ISSUED: August 26, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY

h e b b g e e e f e h ef b e

Haseltine; William A.	Washington	DC
Ruben; Steven M.	Brookeville	MD
Wei; Ying-Fei	Berkeley	CA
Adams; Mark D.	Rockville	MD
Fleischmann; Robert D.	Gaithersburg	MD
Fraser; Claire M.	Potomac	MD
Fuldner; Rebecca A.	Barnesville	MD
Kirkness; Ewen F.	Olney	MD
Rosen; Craig A.	Laytonsville	MD
Vogelstein; Bert	Baltimore	MD
Kinzler; Kenneth W.	Bel Air	MD
Nicolaides; Nicholas C.	Boothwyn	PA
Papadopoulos; Nickolas	Brookline	MA

US-CL-CURRENT: 435/6; 436/94

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw De

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☐ 3. Document ID: US 6482606 B1

L17: Entry 3 of 11 File: USPT Nov 19, 2002

US-PAT-NO: 6482606
DOCUMENT-IDENTIFIER: US 6482606 B1
**** See image for Certificate of Correction ****

TITLE: Human DNA mismatch repair polynucleotides

DATE-ISSUED: November 19, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Adams; Mark D.	North Potomac	MD		
Fleischmann; Robert D.	Washington	DC		
Fraser; Claire M.	Queenstown	MD		
Fuldner; Rebecca A.	Barnesville	MD		
Kirkness; Ewen F.	Washington	DC		
Haseltine; William A.	Washington	DC		
Rosen; Craig A.	Laytonsville	MD		
Ruben; Steve	Olney	MD		
Wei; Ying-Fei	Darnestown	MD		

US-CL-CURRENT: 435/69.1; 435/243, 435/252.3, 435/320.1, 435/325, 435/410, 435/71.1, 435/71.2, 536/23.1, 536/23.2, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw De

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h e b b g e e e f e h ef b e

☐ 4. Document ID: US 6416984 B1

L17: Entry 4 of 11 File: USPT Jul 9, 2002

US-PAT-NO: 6416984
DOCUMENT-IDENTIFIER: US 6416984 B1
**** See image for Certificate of Correction ****

TITLE: Human DNA mismatch repair proteins

DATE-ISSUED: July 9, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Haseltine; William A.	Washington	DC		
Ruben; Steven M.	Olney	MD		
Wei; Ying-Fei	Darnestown	MD		
Adams; Mark D.	North Potomac	MD		
Fleischmann; Robert D.	Gaithersburg	MD		
Fraser; Claire M.	Potomac	MD		
Fuldner; Rebecca A.	Barnesville	MD		
Kirkness; Ewen F.	Olney	MD		
Rosen; Craig A.	Laytonsville	MD		

US-CL-CURRENT: 435/183; 435/195

Full | Title | Citation | Front | Review | Classification | Date | Reference | [illegible] | Claims | KWMC | Draw De

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☐ 5. Document ID: US 6380369 B1

L17: Entry 5 of 11 File: USPT Apr 30, 2002

US-PAT-NO: 6380369
DOCUMENT-IDENTIFIER: US 6380369 B1

TITLE: Human DNA mismatch repair proteins

DATE-ISSUED: April 30, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Adams; Mark D.	North Potomac	MD		
Fleischmann; Robert D.	Gaithersburg	MD		
Fraser; Claire M.	Potomac	MD		
Fuldner; Rebecca A.	Barnesville	MD		
Kirkness; Ewen F.	Olney	MD		
Haseltine; William A.	Washington	DC		
Rosen; Craig A.	Laytonsville	MD		
Ruben; Steve	Olney	MD		
Wei; Ying-Fei	Darnestown	MD		

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US-CL-CURRENT: 536/23.1; 435/6

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw De

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☐ 6. Document ID: US 5801056 A

L17: Entry 6 of 11 File: USPT Sep 1, 1998

US-PAT-NO: 5801056
DOCUMENT-IDENTIFIER: US 5801056 A

TITLE: Nucleic acid encoding HIV-1 tat protein

DATE-ISSUED: September 1, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Haseltine; William Alan	Cambridge	MA		
Rosen; Craig A.	Brookline	MA		
Sodroski; Joseph Gerald	Cambridge	MA		
Wong-Staal; Flossie	San Diego	CA		
Arya; Suresh K.	Gaithersburg	MD		

US-CL-CURRENT: 435/320.1; 536/23.72, 930/221

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw De

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☐ 7. Document ID: US 5800986 A

L17: Entry 7 of 11 File: USPT Sep 1, 1998

US-PAT-NO: 5800986
DOCUMENT-IDENTIFIER: US 5800986 A

TITLE: Assay methods for tat cell lines

DATE-ISSUED: September 1, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Haseltine; William Alan	Cambridge	MA		
Rosen; Craig A.	Brookline	MA		
Sodroski; Joseph Gerald	Cambridge	MA		
Goh; Wei Chun	Somerville	MA		

US-CL-CURRENT: 435/6

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw De

h e b b g e e e f e h ef b e

☐ 8. Document ID: US 5604114 A

L17: Entry 8 of 11 File: USPT Feb 18, 1997

US-PAT-NO: 5604114
DOCUMENT-IDENTIFIER: US 5604114 A

TITLE: Cis-acting repression sequences, cis-acting antirepression sequences, vectors, methods of preparation and use

DATE-ISSUED: February 18, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Haseltine; William A.	Cambridge	MA		
Rosen; Craig A.	Glen Ridge	NJ		
Sodroski; Joseph G.	Cambridge	MA		
Terwilliger; Ernest	Boston	MA		
Goh; Wei C.	Stanford	CA		

US-CL-CURRENT: 435/69.1; 435/320.1, 435/455, 536/23.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | ... | Claims | KWMC | Draw De

☐ 9. Document ID: US 5321124 A

L17: Entry 9 of 11 File: USPT Jun 14, 1994

US-PAT-NO: 5321124
DOCUMENT-IDENTIFIER: US 5321124 A

TITLE: Art (rev) protein of human T-cell leukemia virus

DATE-ISSUED: June 14, 1994

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Haseltine; William A.	Cambridge	MA		
Rosen; Craig A.	Brookline	MA		
Sodroski; Joseph G.	Cambridge	MA		
Goh; Wei C.	Somerville	MA		

US-CL-CURRENT: 530/350; 424/188.1, 424/208.1, 435/235.1, 435/5, 530/395, 930/220, 930/221

Full | Title | Citation | Front | Review | Classification | Date | Reference | ... | Claims | KWMC | Draw De

☐ 10. Document ID: US 4981790 A

h e b b g e e e f e h ef b e

L17: Entry 10 of 11 File: USPT Jan 1, 1991

US-PAT-NO: 4981790
DOCUMENT-IDENTIFIER: US 4981790 A

TITLE: Stable TatIII cell lines, TatIII gene products, and assay methods

DATE-ISSUED: January 1, 1991

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Haseltine; William A.	Cambridge	MA		
Rosen; Craig A.	Brookline	MA		
Sodroski; Joseph G.	Cambridge	MA		
Goh; Wei C.	Somerville	MA		

US-CL-CURRENT: 435/69.1; 435/320.1, 435/357, 435/372, 435/372.2, 435/372.3, 435/465

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw De

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Terms	Documents
L16 and L15	11

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